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USPT	(knock-out or transgenic) near5 mouse near5 cardiotrophin-1	0	L9
USPT	(knock-out or transgenic) near5 (animal or mouse or rat) near5 cardiotrophin-1	0	L8
USPT	cardiotropin-1 near5 gene same (knock-out or transgenic) near5 (animal or mouse or rat)	0	L7
USPT	cardiotropin-1 near5 gene same (knock-out or transgenic) near5 animal or mouse or rat	84735	L6
USPT	(CT-1 or cardiotropin-1) near5 gene same (knock-out or transgenic) near5 animal or mouse or rat	84735	L5
USPT	CT-1 near5 gene same (knock-out or transgenic) near5 animal or mouse or rat	84735	L4
USPT	US 5534615	1	L3
USPT	US 5627073	1	L2
USPT	US 5571893	1	L1

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Terms	Documents
US 5534615	1

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USPT	US 5534615	1	<u>L3</u>
USPT	US 5627073	1	<u>L2</u>
USPT	US 5571893	1	<u>L1</u>

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=> s CT-1 or cardiotrophin adj 1

L1 1445 CT-1 OR CARDIOTROPHIN ADJ 1

=> s CT-1 or cardiotrophin adj 1 (p) animal or mouse or rat

2 FILES SEARCHED...

4 FILES SEARCHED...

L2 6973856 CT-1 OR CARDIOTROPHIN ADJ 1 (P) ANIMAL OR MOUSE OR RAT

=> s transgenic or knock adj out

L3 153725 TRANSGENIC OR KNOCK ADJ OUT

=> s L2 and L3

L4 103951 L2 AND L3

=> s cardiac hypertrophy

L5 18763 CARDIAC HYPERTROPHY

=> s L4 and L5

L6 764 L4 AND L5

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DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS, EMBASE, CAPLUS, SCISEARCH'
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L7 325 DUPLICATE REMOVE L6 (439 DUPLICATES REMOVED)

=> display total ibib abs 1-5 L7

L7 ANSWER 1 OF 325 MEDLINE

ACCESSION NUMBER: 2000273924 MEDLINE

DOCUMENT NUMBER: 20273924

TITLE: Meeting Koch's postulates for calcium signaling in
cardiac hypertrophy [comment].

COMMENT: Comment on: J Clin Invest 2000 May;105(10):1395-406

AUTHOR: Chien K R

CORPORATE SOURCE: University of California at San Diego (UCSD)-Salk Program
in Molecular Medicine, UCSD School of Medicine, Basic
Science Building 0613-C, 9500 Gilman Drive, La Jolla,
California 92093, USA.. kchien@ucsd.edu

SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (2000 May) 105 (10)
1339-42. Ref: 63

Journal code: HS7. ISSN: 0021-9738.

PUB. COUNTRY: United States

Commentary

Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer
Journals

ENTRY MONTH: 200008

ENTRY WEEK: 20000803

L7 ANSWER 2 OF 325 MEDLINE

ACCESSION NUMBER: 2000273931 MEDLINE

DOCUMENT NUMBER: 20273931

TITLE: CaM kinase signaling induces **cardiac**
hypertrophy and activates the MEF2 transcription
factor in vivo [comment].

COMMENT: Comment on: J Clin Invest 2000 May;105(10):1339-42

AUTHOR: Passier R; Zeng H; Frey N; Naya F J; Nicol R L; McKinsey T
A; Overbeek P; Richardson J A; Grant S R; Olson E N
CORPORATE SOURCE: Department of Molecular Biology, The University of Texas
Southwestern Medical Center at Dallas, Dallas, Texas
75235-9148, USA.

SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (2000 May) 105 (10)
1395-406.

Journal code: HS7. ISSN: 0021-9738.

PUB. COUNTRY: United States

Commentary

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer
Journals

ENTRY MONTH: 200008

ENTRY WEEK: 20000803

AB Hypertrophic growth is an adaptive response of the heart to diverse
pathological stimuli and is characterized by cardiomyocyte enlargement,
sarcomere assembly, and activation of a fetal program of cardiac gene
expression. A variety of Ca(2+)-dependent signal transduction pathways
have been implicated in **cardiac hypertrophy**, but
whether these pathways are independent or interdependent and whether
there
is specificity among them are unclear. Previously, we showed that
activation of the Ca(2+)/calmodulin-dependent protein phosphatase
calcineurin or its target transcription factor NFAT3 was sufficient to
evoke myocardial hypertrophy in vivo. Here, we show that activated
Ca(2+)/calmodulin-dependent protein kinases-I and -IV (CaMKI and CaMKIV)
also induce hypertrophic responses in cardiomyocytes in vitro and that

CaMKIV overexpressing **mice** develop **cardiac hypertrophy** with increased left ventricular end-diastolic diameter and decreased fractional shortening. Crossing this **transgenic** line with **mice** expressing a constitutively activated form of NFAT3 revealed synergy between these signaling pathways. We further show that CaMKIV activates the transcription factor MEF2 through a posttranslational mechanism in the hypertrophic heart in vivo. Activated calcineurin is a less efficient activator of MEF2-dependent transcription, suggesting that the calcineurin/NFAT and CaMK/MEF2 pathways act in parallel. These findings identify MEF2 as a downstream target for CaMK signaling in the hypertrophic heart and suggest that the CaMK and

Day : Tuesday
Date:
12/12/2000
Time:
14:06:26



Inventor Information for 09/648183

Inventor Name	City	State/Country
BOTSTEIN, DAVID	BELMONT	CALIFORNIA
GODDARD, AUDREY	SAN FRANCISCO	CALIFORNIA
LAWRENCE, DAVID A.	SAN FRANCISCO	CALIFORNIA
PENNICA, DIANE	BURLINGAME	CALIFORNIA
ROY, MARGARET ANN	SAN FRANCISCO	CALIFORNIA
WOOD, WILLIAM I.	HILLSBOROUGH	CALIFORNIA

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